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☐	L5	human same L4	27
☐	L4	therap$5 same L3	43
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☐	L2	(malignan$5 or cancer or tumor or tumour or neoplas$5)same L1	252
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=> index bioscience medicine

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71 FILES IN THE FILE LIST IN STNINDEX

=> s (arginase or canavanase or (arginine adj amidinase))
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L1 QUE (ARGINASE OR CANAVANASE OR (ARGININE ADJ AMIDINASE))

=> d rank
F1 4660 CAPLUS
F2 3096 BIOSIS
F3 2811 MEDLINE
F4 2115 DGENE
F5 2031 SCISEARCH
F6 1819 EMBASE
F7 1475 TOXCENTER
F8 952 GENBANK
F9 908 USPATFULL
F10 899 PASCAL
F11 708 ESBIOBASE
F12 675 CABA
F13 450 LIFESCI
F14 441 BIOTECHNO
F15 288 AGRICOLA
F16 199 JICST-EPLUS
F17 166 DDFB
F18 166 DRUGB
F19 156 WPIDS
F20 156 WPINDEX

=> file f1-f3, f5-f7, f9-f16

FILE 'CAPLUS' ENTERED AT 18:29:11 ON 03 AUG 2006
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=> s L1
L2 20460 L1

=> S (malignan? or cancer or tumor or tumour or neoplas?)(s)L2
9 FILES SEARCHED...
L3 1236 (MALIGNAN? OR CANCER OR TUMOR OR TUMOUR OR NEOPLAS?)(S) L2

=> s (treat? or prevent? or modulat? or therap?)(s)L3
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8 FILES SEARCHED...
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=> s liver (s)L4
L5 69 LIVER (S) L4

=> dup rem L5
PROCESSING COMPLETED FOR L5
L6 43 DUP REM L5 (26 DUPLICATES REMOVED)

=> d ibib abs L6 1-43

L6 ANSWER 1 OF 43 USPATFULL on STN
ACCESSION NUMBER: 2006:195574 USPATFULL <<LOGINID::20060803>>
TITLE: Albumin fusion proteins
INVENTOR(S): Rosen, Craig A., Laytonsville, MD, UNITED STATES
Haseltine, William A., Washington, DC, UNITED STATES
PATENT ASSIGNEE(S): Human Genome Sciences Inc. (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2006166329	A1	20060727
APPLICATION INFO.:	US 2006-341748	A1	20060130 (11)

RELATED APPLN. INFO.: Continuation of Ser. No. US 2004-816042, filed on 2 Apr 2004, ABANDONED Continuation of Ser. No. WO 2002-US31794, filed on 4 Oct 2002, PENDING

	NUMBER	DATE
PRIORITY INFORMATION:	US 2001-327281P	20011005 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: HUMAN GENOME SCIENCES INC, INTELLECTUAL PROPERTY DEPT., 14200 SHADY GROVE ROAD, ROCKVILLE, MD, 20850, US
NUMBER OF CLAIMS: 19
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 20 Drawing Page(s)
LINE COUNT: 16204
AB The present invention encompasses albumin fusion proteins. Nucleic acid molecules encoding the albumin fusion proteins of the invention are also encompassed by the invention, as are vectors containing these nucleic acids, host cells transformed with these nucleic acids vectors, and methods of making the albumin fusion proteins of the invention and using these nucleic acids, vectors, and/or host cells. Additionally the present invention encompasses pharmaceutical compositions comprising albumin fusion proteins and methods of treating, preventing, or ameliorating diseases, disorders or conditions using albumin fusion proteins of the invention.

L6 ANSWER 2 OF 43 USPATFULL on STN

ACCESSION NUMBER: 2006:137248 USPATFULL <<LOGINID::20060803>>
TITLE: Gene expression profiling for identification monitoring and treatment of multiple sclerosis
INVENTOR(S): Bevilacqua, Michael, Boulder, CO, UNITED STATES
Tryon, Victor V., Woodinville, WA, UNITED STATES
Bankaitis-Davis, Danute, Longmont, CO, UNITED STATES
Siconolfi, Lisa, Westminster, CO, UNITED STATES
Trollinger, David B., Boulder, CO, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2006115826	A1	20060601
APPLICATION INFO.:	US 2005-155930	A1	20050616 (11)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 2003-742458, filed on 19 Dec 2003, PENDING Continuation-in-part of Ser. No. US 2002-291225, filed on 8 Nov 2002, GRANTED, Pat. No. US 6960439 Continuation-in-part of Ser. No. US 2001-821850, filed on 29 Mar 2001, GRANTED, Pat. No. US 6692916 Continuation-in-part of Ser. No. US 2000-605581, filed on 28 Jun 2000, ABANDONED

	NUMBER	DATE
PRIORITY INFORMATION:	US 2002-435257P	20021219 (60)
	US 1999-141542P	19990628 (60)
	US 2000-195522P	20000407 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: BROMBERG & SUNSTEIN LLP, 125 SUMMER STREET, BOSTON, MA, 02110-1618, US
NUMBER OF CLAIMS: 28
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 50 Drawing Page(s)
LINE COUNT: 3338
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A method is provided in various embodiments for determining a profile data set for a subject with multiple sclerosis or inflammatory conditions related to multiple sclerosis based on a sample from the subject, wherein the sample provides a source of RNAs. The method includes using amplification for measuring the amount of RNA corresponding to at least 2 constituents from Table 1. The profile data set comprises the measure of each constituent, and amplification is performed under measurement conditions that are substantially repeatable.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 3 OF 43 USPATFULL on STN
ACCESSION NUMBER: 2006:74111 USPATFULL <<LOGINID::20060803>>
TITLE: Outcome prediction and risk classification in childhood leukemia
INVENTOR(S): Willman, Cheryl L., Albuquerque, NM, UNITED STATES
Helman, Paul, Albuquerque, NM, UNITED STATES
Veroff, Robert, Albuquerque, NM, UNITED STATES
Mosquera-Caro, Monica, Albuquerque, NM, UNITED STATES
Davidson, George S., Albuquerque, NM, UNITED STATES
Martin, Shawn B., Albuquerque, NM, UNITED STATES
Atlas, Susan R., Albuquerque, NM, UNITED STATES
Andries, Erik, Rio Rancho, NM, UNITED STATES
Kang, Huining, Albuquerque, NM, UNITED STATES
Shuster, Jonathan J., Gainesville, FL, UNITED STATES
Wang, Xuefei, Albuquerque, NM, UNITED STATES
Harvey, Richard C., Placitas, NM, UNITED STATES
Haaland, David M., Albuquerque, NM, UNITED STATES
Potter, Jeffrey W., Albuquerque, NM, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2006063156	A1	20060323
APPLICATION INFO.:	US 2003-729895	A1	20031205 (10)

NUMBER DATE

PRIORITY INFORMATION: US 2002-432064P 20021206 (60)
US 2002-432077P 20021206 (60)
US 2002-432078P 20021206 (60)
US 2003-510904P 20031014 (60)
US 2003-510968P 20031014 (60)
US 2003-527610P 20031205 (60)
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: COLEMAN SUDOL SAPONE, P.C., 714 COLORADO AVENUE, BRIDGE PORT, CT, 06605-1601, US
NUMBER OF CLAIMS: 42
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 23 Drawing Page(s)
LINE COUNT: 12227
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Genes and gene expression profiles useful for predicting outcome, risk classification, cytogenetics and/or etiology in pediatric acute lymphoblastic leukemia (ALL). OPAL1 is a novel gene associated with outcome and, along with other newly identified genes, represent a novel therapeutic targets.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 4 OF 43 USPATFULL on STN
ACCESSION NUMBER: 2005:312435 USPATFULL <<LOGINID::20060803>>
TITLE: Molecular genetic profiling of gleason grades 3 and 4/5 prostate cancer
INVENTOR(S): Shekar, Mamatha, Cupertino, CA, UNITED STATES
Zhang, Zhaomei, Sunnyvale, CA, UNITED STATES
Caldwell, Mitchell C., Menlo Park, CA, UNITED STATES
Chen, Zuxiong, Mountain View, CA, UNITED STATES
Fan, Zhenbin, Mountain View, CA, UNITED STATES
McNeal, John E., Oakland, CA, UNITED STATES
Nolley, Rosalie, Menlo Park, CA, UNITED STATES
Stamey, Thomas A., Portola Valley, CA, UNITED STATES
Warrington, Janet A., Los Altos, CA, UNITED STATES
Palma, John F., San Ramon, CA, UNITED STATES
PATENT ASSIGNEE(S): Affymetrix, INC., Santa Clara, CA, UNITED STATES (U.S. corporation)

NUMBER KIND DATE

PATENT INFORMATION: US 2005272052 A1 20051208
APPLICATION INFO.: US 2004-975592 A1 20041027 (10)
RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 2003-411537, filed on 9 Apr 2003, PENDING

NUMBER DATE

PRIORITY INFORMATION: US 2002-371304P 20020409 (60)
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: FISH & NEAVE IP GROUP, ROPES & GRAY, ONE INTERNATIONAL PLACE, BOSTON, MA, 02110, US
NUMBER OF CLAIMS: 25
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 22 Drawing Page(s)
LINE COUNT: 1815
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Many genes are affected in prostate cancers which have not been previously identified. This includes genes that have been up-regulated or down-regulated. Monitoring the expression levels of these genes is useful to identify the existence of prostate cancer. Also, monitoring the expression levels of these genes is useful to predict the effectiveness of treatment, outcome, use of therapeutics, and screening drugs useful for the treatment of prostate cancer.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 5 OF 43 USPATFULL on STN
ACCESSION NUMBER: 2005:280455 USPATFULL <<LOGINID::20060803>>
TITLE: Pharmaceutical preparation and method of treatment of human malignancies with arginine deprivation
INVENTOR(S): Cheng, Ning Man, Hong Kong, CHINA
Leung, Yun Chung, Hong Kong, CHINA
Lo, Wai Hung, Hong Kong, CHINA

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2005244398	A1	20051103
APPLICATION INFO.:	US 2003-518223	A1	20030620 (10)
	WO 2003-GB2665		20030620
			20041215 PCT 371 date

	NUMBER	DATE
PRIORITY INFORMATION:	WO 2002-CN635	20020909
	US 2003-390757P	20020620 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: KNOBBE MARTENS OLSON & BEAR LLP, 2040 MAIN STREET, FOURTEENTH FLOOR, IRVINE, CA, 92614, US
NUMBER OF CLAIMS: 28
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 46 Drawing Page(s)
LINE COUNT: 1684
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention provides an isolated and substantially purified recombinant human arginase having sufficiently high enzymatic activity and stability to maintain Adequate Arginine Depletion in a patient. The present invention also provides a pharmaceutical composition comprising the modified invention enzyme and method for treatment of diseases using the pharmaceutical composition.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 6 OF 43 USPATFULL on STN
ACCESSION NUMBER: 2005:233514 USPATFULL <<LOGINID::20060803>>
TITLE: Methods and apparatuses for diagnosing AML and MDS
INVENTOR(S): Burczynski, Michael E., Swampscott, MA, UNITED STATES
Dorner, Andrew J., Lexington, MA, UNITED STATES
Twine, Natalie C., Goffstown, NH, UNITED STATES
Trepicchio, William L., Andover, MA, UNITED STATES
Stover, Jennifer, Topsfield, MA, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2005202451	A1	20050915
APPLICATION INFO.:	US 2004-834114	A1	20040429 (10)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2003-466055P	20030429 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: NIXON PEABODY, LLP, 401 9TH STREET, NW, SUITE 900, WASHINGTON, DC, 20004-2128, US
NUMBER OF CLAIMS: 20
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 3 Drawing Page(s)
LINE COUNT: 8813
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Methods, systems and equipment for diagnosing or monitoring the progression or treatment of AML or MDS. This invention identifies a plurality of AML or MDS disease genes which are differentially expressed in bone marrow cells of AML or MDS patients as compared to disease-free humans. These AML or MDS disease genes can be used as molecular markers

for detecting the presence or absence of AML or MDS. These genes can also be used for the early identification of MDS patients who eventually progress to AML.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 7 OF 43 USPATFULL on STN
ACCESSION NUMBER: 2005:99567 USPATFULL <<LOGINID::20060803>>
TITLE: Methods of use of inhibitors of phosphodiesterases and modulators of nitric oxide, reactive oxygen species, and metalloproteinases in the treatment of peyronie's disease, arteriosclerosis and other fibrotic diseases
INVENTOR(S): Gonzalez-Cadavid, Nestor F., Pasadena, CA, UNITED STATES
Rajfer, Jacob, Rolling Hills Estates, CA, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2005085486	A1	20050421
APPLICATION INFO.:	US 2004-779069	A1	20040213 (10)

	NUMBER	DATE
PRIORITY INFORMATION:	WO 2003-US33400	20031021

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: BLAKELY SOKOLOFF TAYLOR & ZAFMAN, 12400 WILSHIRE BOULEVARD, SEVENTH FLOOR, LOS ANGELES, CA, 90025-1030, US
NUMBER OF CLAIMS: 29
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 21 Drawing Page(s)
LINE COUNT: 4042
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present methods and compositions are of use for treatment of conditions involving fibrosis, such as Peyronie's disease plaque, penile corporal fibrosis, penile veno-occlusive dysfunction, Dupuytren's disease nodules, vaginal fibrosis, clitoral fibrosis, female sexual arousal disorder, abnormal wound healing, keloid formation, general fibrosis of the kidney, bladder, prostate, skin, liver, lung, heart, intestines or any other localized or generalized fibrotic condition, vascular fibrosis, arterial intima hyperplasia, atherosclerosis, arteriosclerosis, restenosis, cardiac hypertrophy, hypertension or any condition characterized by excessive fibroblast or smooth muscle cell proliferation or deposition of collagen and extracellular matrix in the blood vessels and/or heart. In certain embodiments, the compositions may comprise a PDE-4 inhibitor, a PDE-5 inhibitor, a compound that elevates cGMP and/or PKG, a stimulator of guanylyl cyclase and/or PKG, a combination of a compound that elevates cGMP, PKG or NO with an antioxidant that decreases ROS, or a compound that increases MMP activity.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 8 OF 43 USPATFULL on STN
ACCESSION NUMBER: 2005:69957 USPATFULL <<LOGINID::20060803>>
TITLE: Systems and methods for characterizing a biological condition or agent using precision gene expression profiles
INVENTOR(S): Bevilacqua, Michael P., Boulder, CO, UNITED STATES
Bankaitis-Davis, Danute M., Longmont, CO, UNITED STATES
Cheronis, John C., Conifer, CO, UNITED STATES
Tryon, Victor, Loveland, CO, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2005060101	A1	20050317
APPLICATION INFO.:	US 2003-742458	A1	20031219 (10)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 2002-291225, filed on 8 Nov 2002, PENDING Continuation-in-part of Ser. No.

US 2001-821850, filed on 29 Mar 2001, GRANTED, Pat. No.
US 6692916 Continuation-in-part of Ser. No. US
2000-605581, filed on 28 Jun 2000, ABANDONED

NUMBER DATE

PRIORITY INFORMATION: US 2002-435257P 20021219 (60)
US 1999-141542P 19990628 (60)
US 2000-195522P 20000407 (60)
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: HEWLETT PACKARD COMPANY, P O BOX 272400, 3404 E. HARMONY ROAD, INTELLECTUAL PROPERTY ADMINISTRATION, FORT COLLINS, CO, 80527-2400
NUMBER OF CLAIMS: 261
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 49 Drawing Page(s)
LINE COUNT: 5083
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A method is provided in various embodiments for determining a profile data set for a subject with infectious disease or inflammatory conditions related to infectious disease based on a sample from the subject, wherein the sample provides a source of RNAs. The method includes using amplification for measuring the amount of RNA corresponding to at least 2 constituents from Table 1. The profile data set comprises the measure of each constituent, and amplification is performed under measurement conditions that are substantially repeatable.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 9 OF 43 USPATFULL on STN
ACCESSION NUMBER: 2005:43296 USPATFULL <<LOGINID::20060803>>
TITLE: Albumin fusion proteins
INVENTOR(S): Rosen, Craig A., Laytonsville, MD, UNITED STATES
Haseltine, William A., Washington, DC, UNITED STATES

NUMBER KIND DATE

PATENT INFORMATION: US 2005037022 A1 20050217
APPLICATION INFO.: US 2004-816042 A1 20040402 (10)
RELATED APPLN. INFO.: Continuation of Ser. No. WO 2002-US31794, filed on 4 Oct 2002, PENDING

NUMBER DATE

PRIORITY INFORMATION: US 2001-327281P 20011005 (60)
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: HUMAN GENOME SCIENCES INC, INTELLECTUAL PROPERTY DEPT., 14200 SHADY GROVE ROAD, ROCKVILLE, MD, 20850
NUMBER OF CLAIMS: 29
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 18 Drawing Page(s)
LINE COUNT: 17090
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention encompasses albumin fusion proteins. Nucleic acid molecules encoding the albumin fusion proteins of the invention are also encompassed by the invention, as are vectors containing these nucleic acids, host cells transformed with these nucleic acids vectors, and methods of making the albumin fusion proteins of the invention and using these nucleic acids, vectors, and/or host cells. Additionally the present invention encompasses pharmaceutical compositions comprising albumin fusion proteins and methods of treating, preventing, or ameliorating diseases, disordrs or conditions using albumin fusion proteins of the invention.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 10 OF 43 CAPLUS COPYRIGHT 2006 ACS on STN DUPLICATE 1

ACCESSION NUMBER: 2005:462751 CAPLUS <<LOGINID::20060803>>
DOCUMENT NUMBER: 143:205885
TITLE: Remission of hepatocellular carcinoma with arginine depletion induced by systemic release of endogenous hepatic arginase due to transhepatic arterial embolisation, augmented by high-dose insulin: arginase as a potential drug candidate for hepatocellular carcinoma
AUTHOR(S): Cheng, P. N. M.; Leung, Y. C.; Lo, W. H.; Tsui, S. M.; Lam, K. C.
CORPORATE SOURCE: Department of Applied Biology and Chemical Technology, The Hong Kong Polytechnic University, Hong Kong, Kowloon, Hung Hom, Peop. Rep. China
SOURCE: Cancer Letters (Amsterdam, Netherlands) (2005), 224(1), 67-80
CODEN: CALEDQ; ISSN: 0304-3835
PUBLISHER: Elsevier B.V.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Hepatocellular carcinoma (HCC) is auxotrophic for the semi-essential amino acid arginine, depletion of which leads to tumor death. In humans, arginine is not an essential amino acid since many adult somatic cells can re-synthesize it from other sources, such as citrulline. Enzymes capable of depleting arginine in vitro include the urea cycle enzyme arginase, which is found in abundance in human liver. For over three decades, arginase has not been considered as a potential drug candidate because of its low substrate affinity, short circulatory half-life and sub-optimal enzymic activity at physiol. pH, though its in vitro antitumor activities in certain tumors have been amply reported. Arginine deiminase, a bacterial enzyme from Mycoplasma hominus has been shown to induce HCC remission through the mechanism of arginine depletion. We report here an innovative ***treatment*** approach for the ***treatment*** of locally advanced and metastatic HCC with transhepatic arterial embolization (TAE) of the ***liver*** ***tumor*** with lipiodol and gel foam as a means of inducing a leakage of hepatic ***arginase*** from the ***liver*** into the circulation. Hepatic arginase released into the systemic circulation rapidly depleted plasma arginine. High-dose insulin was included to induce a state of hypoaminoacidemia to augment arginine depletion. With this protocol, we have treated seven patients with locally advanced and/or metastatic HCC. Five patients achieved arginine depletion, ranging from 0 to 20 .mu.M (normal plasma level 100-120 .mu.M); all had varying degrees of tumor remission in their primary tumors and extra-hepatic sites in the lymph nodes, lungs and bones, suggesting systemic anti-cancer effect of arginine depletion. The two non-responders did not show significant redn. in plasma arginine. Based on our findings, we propose that the urea cycle enzyme, arginase, is a good drug candidate for the treatment of HCC.
REFERENCE COUNT: 32 THERE ARE 32 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 11 OF 43 USPATFULL on STN
ACCESSION NUMBER: 2004:320664 USPATFULL <<LOGINID::20060803>>
TITLE: Pharmaceutical preparation for the treatment and diagnosis of tumors and method for the preparation of the lipid free fraction of blood plasma
INVENTOR(S): Bertha, Andras, Budapest, HUNGARY

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2004253317	A1	20041216
APPLICATION INFO.:	US 2003-332440	A1	20030108 (10)
	WO 2001-HU78		20010710

	NUMBER	DATE
PRIORITY INFORMATION:	HU 2000-P2597	20000710

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: ANTHONY H. HANDAL, KIRKPATRICK & LOCKHART, LLP, 599 LEXINGTON AVENUE, 31ST FLOOR, NEW YORK, NY, 10022-6030

NUMBER OF CLAIMS: 8
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 18 Drawing Page(s)
LINE COUNT: 809
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Pharmaceutical preparation for the treatment and follow-up care of tumors that comprises the blood plasma or predetermined blood plasma components of equidae animals with pair number of fingers, preferably cattle being not endangered lethally by leucosis. The predetermined fraction is the material bovin 40 and/or bovin 300 constituting the difference detectable by electrophoresis between the lipid-free fraction taken from a cattle having leucosis and the lipid-free fraction taken from a healthy cattle.

In the separation method of the required fraction the initial blood is optionally treated by an anti-coagulant and the corpuscles are separated therefrom, the plasma fraction is treated by a first organic solvent, then a surfactant material composed of fine grains is added thereto, the liquid is mixed and the lipid-free fraction bound to the grains is separated from the liquid components by centrifugation, and the separated fraction is brought again into a solution.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 12 OF 43 USPATFULL on STN
ACCESSION NUMBER: 2004:306537 USPATFULL <<LOGINID::20060803>>
TITLE: Matrices for drug delivery and methods for making and using the same
INVENTOR(S): Babich, John W., North Scituate, MA, UNITED STATES
Zubieta, Jon, Syracuse, NY, UNITED STATES
Bonavia, Grant, Kensington, MD, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2004241205	A1	20041202
	US 7052913	B2	20060530
APPLICATION INFO.:	US 2004-838423	A1	20040504 (10)

RELATED APPLN. INFO.: Continuation of Ser. No. US 2002-77475, filed on 15 Feb 2002, ABANDONED Continuation of Ser. No. US 2000-503438, filed on 14 Feb 2000, GRANTED, Pat. No. US 6395299

	NUMBER	DATE
PRIORITY INFORMATION:	US 1999-119828P	19990212 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: FOLEY HOAG, LLP, PATENT GROUP, WORLD TRADE CENTER WEST, 155 SEAPORT BLVD, BOSTON, MA, 02110
NUMBER OF CLAIMS: 157
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 13 Drawing Page(s)
LINE COUNT: 4315
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB In one aspect, biocompatible matrices such as sol-gels encapsulating a reaction center may be administered to a subject for conversion of prodrugs into biologically active agents. In certain embodiments, the biocompatible matrices of the present invention are sol-gels. In one embodiment, the enzyme L-amino acid decarboxylase is encapsulated and implanted in the brain to convert L-dopa to dopamine for treatment of Parkinson's disease.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 13 OF 43 USPATFULL on STN
ACCESSION NUMBER: 2004:83210 USPATFULL <<LOGINID::20060803>>
TITLE: Compositions for inhibiting arginase activity
INVENTOR(S): Christianson, David, Media, PA, UNITED STATES
Baggio, Ricky, Waltham, MA, UNITED STATES
Elbaum, Daniel, Newton, MA, UNITED STATES

NUMBER KIND DATE

PATENT INFORMATION: US 2004063666 A1 20040401
APPLICATION INFO.: US 2003-661965 A1 20030912 (10)
RELATED APPLN. INFO.: Continuation of Ser. No. US 2002-53939, filed on 23 Jan 2002, PENDING Division of Ser. No. US 2000-545737, filed on 10 Apr 2000, GRANTED, Pat. No. US 6387890 Continuation-in-part of Ser. No. WO 1998-US21430, filed on 9 Oct 1998, PENDING
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: DUANE MORRIS, LLP, ATTN: WILLIAM H. MURRAY, ONE LIBERTY PLACE, 1650 MARKET STREET, PHILADELPHIA, PA, 19103-7396
NUMBER OF CLAIMS: 5
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 34 Drawing Page(s)
LINE COUNT: 3205
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Compositions and methods for inhibiting arginase activity, including arginase activity in a mammal, are provided. Methods of making the compositions of the invention are also provided as are methods of using the compositions therapeutically. The compositions described herein are useful for alleviating or inhibiting a variety of arinase- and NO synthase-related disorders, including heart diseae, gastrointestinal motility disorders, and penile erectile dysfunction in humans.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 14 OF 43 USPATFULL on STN
ACCESSION NUMBER: 2004:76149 USPATFULL <<LOGINID::20060803>>
TITLE: Modulation of the immune response through the manipulation of arginine levels
INVENTOR(S): Ochoa, Augusto C., New Orleans, LA, UNITED STATES
Ochoa, Juan B., Pittsburgh, PA, UNITED STATES
Popescu, Mircea, Plainsboro, NJ, UNITED STATES
Zea, Arnold H., Metairie, LA, UNITED STATES
Rodriguez, Paulo C., Metairie, LA, UNITED STATES
PATENT ASSIGNEE(S): LSU Medical Center (U.S. corporation)

NUMBER KIND DATE

PATENT INFORMATION: US 2004057926 A1 20040325
APPLICATION INFO.: US 2003-386131 A1 20030312 (10)

NUMBER DATE

PRIORITY INFORMATION: US 2002-363366P 20020312 (60)
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: FOLEY AND LARDNER, SUITE 500, 3000 K STREET NW, WASHINGTON, DC, 20007
NUMBER OF CLAIMS: 20
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 30 Drawing Page(s)
LINE COUNT: 3404
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention provides methods and compositions for modulating an immune response by controlling the level of arginase available to a cell, tissue or system. An immune response can be enhanced or depressed by altering the amount of arginine available to a cell, tissue or system through the manipulation of localized or systemic arginine levels using substances which provide arginine to the body and enzymes which break down arginine, such as arginase and nitric oxide synthase. Increasing or decreasing an immune response according to the present invention provides therapeutic treatments for a variety of conditions and diseases. The present invention also provides clinical methods and kits which can measure the strength or resistance to an immune response in a cell, tissue or system based upon the amount of available arginine and enzymes which break down arginine.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 15 OF 43 USPATFULL on STN
ACCESSION NUMBER: 2004:38612 USPATFULL <<LOGINID::20060803>>
TITLE: Molecular genetic profiling of gleason grades 3 and 4/5 prostate cancer
INVENTOR(S): Mahadevappa, Mamatha, Fremont, CA, UNITED STATES
Zhang, Zhaomei, Sunnyvale, CA, UNITED STATES
Warrington, Janet A., Los Altos, CA, UNITED STATES
Palma, John F., San Ramon, CA, UNITED STATES
Caldwell, Mitchell C., Menlo Park, CA, UNITED STATES
Chen, Zuxiong, Sunnyvale, CA, UNITED STATES
Fan, Zhenbin, Mountain View, CA, UNITED STATES
McNeal, John E., Oakland, CA, UNITED STATES
Nolley, Rosalie, Menlo Park, CA, UNITED STATES
Stamey, Thomas A., Menlo Park, CA, UNITED STATES
PATENT ASSIGNEE(S): Affymetrix, INC., Santa Clara, CA, UNITED STATES (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2004029151	A1	20040212
APPLICATION INFO.:	US 2003-411537	A1	20030409 (10)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2002-371304P	20020409 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: AFFYMETRIX, INC, ATTN: CHIEF IP COUNSEL, LEGAL DEPT., 3380 CENTRAL EXPRESSWAY, SANTA CLARA, CA, 95051
NUMBER OF CLAIMS: 25
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 22 Drawing Page(s)
LINE COUNT: 1825
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Many genes are affected in prostate cancers which have not been previously identified. This includes genes that have been up-regulated or down-regulated. Monitoring the expression levels of these genes is useful to identify the existence of prostate cancer. Also, monitoring the expression levels of these genes is useful to predict the effectiveness of treatment, outcome, use of therapeutics, and screening drugs useful for the treatment of prostate cancer.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 16 OF 43 USPATFULL on STN
ACCESSION NUMBER: 2004:66006 USPATFULL <<LOGINID::20060803>>
TITLE: DNA array sequence selection
INVENTOR(S): Lorenz, Matthias, Bethesda, MD, United States
PATENT ASSIGNEE(S): The United States of America as represented by the Department of Health and Human Services, Washington, DC, United States (U.S. government)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6706867	B1	20040316
APPLICATION INFO.:	US 2000-741238		20001219 (9)

DOCUMENT TYPE: Utility
FILE SEGMENT: GRANTED
PRIMARY EXAMINER: Horlick, Kenneth R.
ASSISTANT EXAMINER: Wilder, Cynthia
LEGAL REPRESENTATIVE: Leydig, Voit & Mayer, Ltd.
NUMBER OF CLAIMS: 8
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 3 Drawing Figure(s); 29 Drawing Page(s)
LINE COUNT: 23532
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention provides methods and compositions for the

construction of custom cDNA microarrays. In particular, the methods involve the selection of relevant clusters based on knowledge and expression patterns using public database information and the identification of the best representative cDNA clones within the selected cluster. The methods facilitate the construction of custom microarrays suitable for use in any biotechnological art. In preferred embodiments, the present invention provides the the ImmunoChip.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 17 OF 43 USPATFULL on STN
ACCESSION NUMBER: 2004:4504 USPATFULL <<LOGINID::20060803>>
TITLE: Tumor necrosis factor receptor 2
INVENTOR(S): Stanton, Jr., Vincent P., Belmont, MA, United States
PATENT ASSIGNEE(S): Nuvelo, Inc., Sunnyvale, CA, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6673908	B1	20040106
APPLICATION INFO.:	US 2001-968455		20011001 (9)

RELATED APPLN. INFO.: Division of Ser. No. US 2000-649035, filed on 25 Aug 2000 Continuation-in-part of Ser. No. US 2000-590749, filed on 8 Jun 2000, now abandoned Continuation-in-part of Ser. No. US 2000-495780, filed on 1 Feb 2000, now abandoned Continuation-in-part of Ser. No. US 2000-492712, filed on 27 Jan 2000, now abandoned Continuation-in-part of Ser. No. WO 2000-US1392, filed on 20 Jan 2000 Continuation-in-part of Ser. No. US 968455 Continuation-in-part of Ser. No. US 1999-451252, filed on 29 Nov 1999, now abandoned Continuation-in-part of Ser. No. US 1999-427835, filed on 26 Oct 1999, now abandoned Continuation-in-part of Ser. No. US 1999-414330, filed on 6 Oct 1999, now abandoned Continuation-in-part of Ser. No. US 1999-389993, filed on 3 Sep 1999, now abandoned Continuation-in-part of Ser. No. US 1999-370841, filed on 9 Aug 1999, now abandoned Continuation-in-part of Ser. No. US 1999-300747, filed on 26 Apr 1999, now abandoned

	NUMBER	DATE
PRIORITY INFORMATION:	US 1999-131334P	19990426 (60)
	US 1999-131191P	19990426 (60)
	US 1999-121047P	19990222 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: GRANTED
PRIMARY EXAMINER: Benzion, Gary
ASSISTANT EXAMINER: Chakrabarti, Arun Kr.
LEGAL REPRESENTATIVE: Fish & Richardson P.C.
NUMBER OF CLAIMS: 10
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 0 Drawing Figure(s); 0 Drawing Page(s)
LINE COUNT: 17463
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present disclosure describes the use of genetic variance information for genes involved in inflammatory or immunologic disease, disorder, or dysfunction. The variance information is indicative of the expected response of a patient to a method of treatment. Methods of determining relevant variance information and additional methods of using such variance information are also described.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 18 OF 43 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2004:3046 CAPLUS <<LOGINID::20060803>>
DOCUMENT NUMBER: 140:72158
TITLE: Protein and cDNA sequences of human arginase I and use for treatment of human malignancies with arginine

deprivation
INVENTOR(S): Cheng, Ningman; Leung, Yunchung; Lo, Waihung
PATENT ASSIGNEE(S): Bio-Cancer Treatment International Limited, Peop. Rep. China
SOURCE: PCT Int. Appl., 93 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2004001048	A1	20031231	WO 2002-CN635	20020909
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
HK 1053577	A2	20031010	HK 2002-106593	20020906
AU 2002325782	A1	20040106	AU 2002-325782	20020909
WO 2004000349	A1	20031231	WO 2003-GB2665	20030620
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
AU 2003250371	A1	20040106	AU 2003-250371	20030620
EP 1517699	A1	20050330	EP 2003-760800	20030620
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
JP 2005538709	T2	20051222	JP 2004-530914	20030620
US 2005244398	A1	20051103	US 2004-518223	20041215

PRIORITY APPLN. INFO.: US 2002-390757P P 20020620
WO 2002-CN635 W 20020909
WO 2003-GB2665 W 20030620

AB The present invention provides protein and cDNA sequences of human arginase I having sufficiently high enzymic activity and stability to maintain AAD in a patient. The present invention also provides a pharmaceutical compn. comprising the modified invention enzyme and method for treatment of diseases using the invention pharmaceutical compn.

REFERENCE COUNT: 7 THERE ARE 7 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 19 OF 43 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2004:2726 CAPLUS <<LOGINID::20060803>>
DOCUMENT NUMBER: 140:70997
TITLE: Human arginase I manufactured in a bacterial host for treatment of cancer by arginine deprivation
INVENTOR(S): Cheng, Ning Man; Leung, Yun Chung; Lo, Wai Hung
PATENT ASSIGNEE(S): Bio-Cancer Treatment International Limited, Peop. Rep. China
SOURCE: PCT Int. Appl., 86 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE

WO 2004000349	A1	20031231	WO 2003-GB2665	20030620

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

WO 2004001048	A1	20031231	WO 2002-CN635	20020909

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

AU 2003250371	A1	20040106	AU 2003-250371	20030620
EP 1517699	A1	20050330	EP 2003-760800	20030620

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK

JP 2005538709	T2	20051222	JP 2004-530914	20030620
US 2005244398	A1	20051103	US 2004-518223	20041215

PRIORITY APPLN. INFO.: US 2002-390757P P 20020620
WO 2002-CN635 A 20020909
WO 2003-GB2665 W 20030620

AB Human arginase I is manufd. using a bacterial expression host for use in the treatment of cancers by inducing arginine depletion in a patient. The enzyme may be stabilized, e.g. by conjugation with water sol. polymers, for therapeutic use. It is preferably used in combination with other drugs, e.g. insulin, that prevent proteolysis and the recycling of protein arginine. The present invention also provides a pharmaceutical compn. comprising the modified invention enzyme and method for treatment of diseases using the pharmaceutical compn.

REFERENCE COUNT: 6 THERE ARE 6 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 20 OF 43 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2003:610191 CAPLUS <<LOGINID::20060803>>
DOCUMENT NUMBER: 139:154917
TITLE: Therapeutic composition for treatment of cancer by arginine depletion
INVENTOR(S): Tepic, Slobodan
PATENT ASSIGNEE(S): Cancer Treatments International, Switz.; Orzechowski, Karen Lee
SOURCE: PCT Int. Appl., 43 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003063780	A2	20030807	WO 2003-US2342	20030127
WO 2003063780	A3	20041111		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES,

FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG
EP 1499342 A2 20050126 EP 2003-735013 20030127
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK
PRIORITY APPLN. INFO.: US 2002-350971P P 20020125
WO 2003-US2342 W 20030127
AB A therapeutic compn. and a method for the treatment of cancer by depletion of arginine without systemic complications comprising an arginine decompg. enzyme and protein breakdown inhibitors, a nitric oxide donor, a pressor peptide, and prostacyclin. The compn. may further include an amino acid mixt. lacking arginine, an antidote for cyanide, blood plasma or its derivs., and/or a prepn. of arginine. The arginine decompg. enzyme may modified to increase circulation half-life and can be type I liver arginase, or type II of human or animal, partially purified, or recombinant, or even bacterial origin. It may be administered as a drug or released from the patient's own tissue. Endogenous prodn. of arginine, particularly via so-called intestinal-kidney axis, can be beneficially inhibited at several enzymic steps, allowing for deeper redns. of circulating arginine. Different components of the compn. may be administered sep., or in suitable mixts., allowing for needed adjustments during the treatment.

L6 ANSWER 21 OF 43 USPATFULL on STN
ACCESSION NUMBER: 2003:318678 USPATFULL <<LOGINID::20060803>>
TITLE: Pre-and post therapy gene expression profiling to identify drug targets
INVENTOR(S): Evans, William Edward, Cordova, TN, UNITED STATES
Relling, Mary V., Cordova, TN, UNITED STATES
PATENT ASSIGNEE(S): St. Jude Children's Research Hospital, Inc. (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003224422	A1	20031204
APPLICATION INFO.:	US 2003-407790	A1	20030404 (10)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2002-370835P	20020408 (60)
	US 2003-449893P	20030225 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: ALSTON & BIRD LLP, BANK OF AMERICA PLAZA, 101 SOUTH TRYON STREET, SUITE 4000, CHARLOTTE, NC, 28280-4000
NUMBER OF CLAIMS: 48
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 2 Drawing Page(s)
LINE COUNT: 2478
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A general method for identifying biological targets for improving currently available therapies is provided. Target genes and their expression products are identified based on their response to therapy as determined through pre- and post-therapy expression profiles. In another aspect, differences in expression profiles between responsive and nonresponsive patients are taken into account to identify potential new targets for the development of novel medications or treatments. The invention also provides methods for comparing therapies to predict which will have the best therapeutic efficacy and/or the least potential deleterious. The methods taught are specifically applied to identify targets for improving treatment of acute lymphoblastic leukemia.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 22 OF 43 USPATFULL on STN
ACCESSION NUMBER: 2003:119753 USPATFULL <<LOGINID::20060803>>
TITLE: Matrices for drug delivery and methods for making and using the same
INVENTOR(S): Babich, John W., North Scituate, MA, UNITED STATES
Zubieta, Jon, Syracuse, NY, UNITED STATES

Bonavia, Grant, Kensington, MD, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003082238	A1	20030501
APPLICATION INFO.:	US 2002-77475	A1	20020215 (10)

RELATED APPLN. INFO.: Continuation of Ser. No. US 2000-503438, filed on 14 Feb 2000, GRANTED, Pat. No. US 6395299

	NUMBER	DATE
PRIORITY INFORMATION:	US 1999-119828P	19990212 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: FOLEY, HOAG & ELIOT, LLP, PATENT GROUP, ONE POST OFFICE SQUARE, BOSTON, MA, 02109
NUMBER OF CLAIMS: 138
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 13 Drawing Page(s)
LINE COUNT: 4259
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB In one aspect, biocompatible matrices such as sol-gels encapsulating a reaction center may be administered to a subject for conversion of prodrugs into biologically active agents. In certain embodiments, the biocompatible matrices of the present invention are sol-gels. In one embodiment, the enzyme L-amino acid decarboxylase is encapsulated and implanted in the brain to convert L-dopa to dopamine for treatment of Parkinson's disease.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 23 OF 43 USPATFULL on STN
ACCESSION NUMBER: 2003:51571 USPATFULL <<LOGINID::20060803>>
TITLE: Compositions and methods for inhibiting arginase activity
INVENTOR(S): Christianson, David, Media, PA, UNITED STATES
Baggio, Ricky, Waltham, MA, UNITED STATES
Elbaum, Daniel, Newton, MA, UNITED STATES
PATENT ASSIGNEE(S): The Trustees of the University of Pennsylvania, Philadelphia, PA, UNITED STATES, 19104-3147 (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003036529	A1	20030220
	US 6723710	B2	20040420
APPLICATION INFO.:	US 2002-53939	A1	20020123 (10)

RELATED APPLN. INFO.: Division of Ser. No. US 2000-545737, filed on 10 Apr 2000, GRANTED, Pat. No. US 6387890 Continuation-in-part of Ser. No. WO 1998-US21430, filed on 9 Oct 1998, UNKNOWN

	NUMBER	DATE
PRIORITY INFORMATION:	US 1997-61607P	19971010 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: AKIN, GUMP, STRAUSS, HAUER & FELD, L.L.P., ONE COMMERCE SQUARE, 2005 MARKET STREET, SUITE 2200, PHILADELPHIA, PA, 19103
NUMBER OF CLAIMS: 5
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 34 Drawing Page(s)
LINE COUNT: 3194
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Compositions and methods for inhibiting arginase activity, including arginase activity in a mammal, are provided. Methods of making the compositions of the invention are also provided as are methods of using the compositions therapeutically. The compositions described herein are useful for alleviating or inhibiting a variety of arinase- and NO

synthase-related disorders, including heart diseae, gastrointestinal motility disorders, and penile erectile dysfunction in humans.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 24 OF 43 USPATFULL on STN
ACCESSION NUMBER: 2002:109029 USPATFULL <<LOGINID::20060803>>
TITLE: Compositions and methods for inhibiting arginase activity
INVENTOR(S): Christianson, David, Media, PA, United States
Baggio, Ricky, Waltham, MA, United States
Elbaum, Daniel, Newton, MA, United States
PATENT ASSIGNEE(S): Trustees of the University of Pennsylvania, Philadelphia, PA, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6387890	B1	20020514
APPLICATION INFO.:	US 2000-545737		20000410 (9)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. WO 1998-US21430, filed on 9 Oct 1998

	NUMBER	DATE
PRIORITY INFORMATION:	US 1997-61607P	19971010 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: GRANTED
PRIMARY EXAMINER: Vollano, Jean F.
LEGAL REPRESENTATIVE: Akin, Gump, Strauss, Hauer & Feld, L.L.P.
NUMBER OF CLAIMS: 26
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 47 Drawing Figure(s); 34 Drawing Page(s)
LINE COUNT: 3302
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Compositions and methods for inhibiting arginase activity, including arginase activity in a mammal, are provided. Methods of making the compositions of the invention are also provided as are methods of using the compositions therapeutically. The compositions described herein are useful for alleviating or inhibiting a variety of arinase- and NO synthase-related disorders, including heart diseae, gastrointestinal motility disorders, and penile erectile dysfunction in humans.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 25 OF 43 CAPLUS COPYRIGHT 2006 ACS on STN DUPLICATE 2
ACCESSION NUMBER: 2002:815788 CAPLUS <<LOGINID::20060803>>
DOCUMENT NUMBER: 138:51834
TITLE: Arginase activity is inhibited by L-NAME, both in vitro and in vivo
AUTHOR(S): Reisser, Daniele; Onier-Cherix, Nathalie; Jeannin, Jean-Francois
CORPORATE SOURCE: Faculty of Medicine and Pharmacy, Cancer Immunotherapy Research Laboratory, Ecole Pratique des Hautes Etudes, Inserm U517, Dijon, 21079, Fr.
SOURCE: Journal of Enzyme Inhibition and Medicinal Chemistry (2002), 17(4), 267-270
CODEN: JEIMAZ; ISSN: 1475-6366
PUBLISHER: Taylor & Francis Ltd.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The present study investigated the ability of the arginine analog L-NAME (N.omega.-Nitro-L-arginine Me ester) to modulate the activity of arginase. L-NAME inhibited the activity of arginase in lysates from rat colon cancer cells and liver. It also inhibited the arginase activity of tumor cells in culture. Furthermore, in vivo ***treatment*** of rats with L-NAME inhibited ***arginase*** activity in ***tumor*** nodules and ***liver*** , and the effect persisted after ***treatment*** ceased. The effect of L-NAME on arginase requires consideration when it is used in vivo in animal models with the aim of inhibiting endothelial NO-synthase, another enzyme using arginine as substrate.

REFERENCE COUNT: 19 THERE ARE 19 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 26 OF 43 PASCAL COPYRIGHT 2006 INIST-CNRS. ALL RIGHTS RESERVED. on STN DUPLICATE 3
ACCESSION NUMBER: 2001-0161046 PASCAL <<LOGINID::20060803>>
COPYRIGHT NOTICE: Copyright .COPYRGT. 2001 INIST-CNRS. All rights reserved.
TITLE (IN ENGLISH): Macrophage arginase promotes tumor cell growth and suppresses nitric oxide-mediated tumor cytotoxicity
AUTHOR: CHANG Chiung-I; LIAO James C.; KUO Lih
CORPORATE SOURCE: Department of Medical Physiology, Cardiovascular Research Institute. Texas A&M University System Health Science Center, College Station. Texas 77843-1114, United States; Department of Chemical Engineering, University of California, Los Angeles, California 90095-1592, United States
SOURCE: Cancer research : (Baltimore), (2001), 61(3), 1100-1106, 37 refs.
ISSN: 0008-5472 CODEN: CNREA8
DOCUMENT TYPE: Journal
BIBLIOGRAPHIC LEVEL: Analytic
COUNTRY: United States
LANGUAGE: English
AVAILABILITY: INIST-5088, 354000098658670540
AN 2001-0161046 PASCAL <<LOGINID::20060803>>
CP Copyright .COPYRGT. 2001 INIST-CNRS. All rights reserved.
AB Macrophages use L-arginine to synthesize nitric oxide (NO) and polyamines through the inducible NO synthase (iNOS) and ***arginase*** , respectively. The released NO contributes to the tumoricidal activity of macrophages, whereas polyamines may promote the growth of ***tumor*** cells. Both the tumoricidal and growth-promoting activities from macrophages have been reported; however, the underlying mechanisms for switching between this dual function of macrophages remain unclear. Here, we test the hypothesis that ***arginase*** participates in the switching between the cytotoxic and growth-promoting activities of macrophages toward ***tumor*** cells. To alter ***arginase*** activity in macrophages, cells (murine macrophage cell line J774A.1) were transfected with the rat ***liver*** ***arginase*** gene or ***treated*** with an ***arginase*** inhibitor, L-norvaline. The effects of macrophage ***arginase*** activity on the growth-promoting and cytotoxic activities of macrophages toward breast ***tumor*** cells (ZR-75-1) were investigated in a coculture system. The results demonstrated that overexpression of ***arginase*** in macrophages enhanced L-ornithine and putrescine production and consequently promoted ***tumor*** cell proliferation. This proliferative effect was down-regulated by the ***arginase*** inhibitor L-norvaline. Furthermore, increases in ***arginase*** activity also attenuated NO production by the lipopolysaccharide-activated macrophages and thus reduced the cytotoxic effect on cocultured ***tumor*** cells. Inhibiting ***arginase*** activity by L-norvaline effectively reversed the suppression of NO-mediated ***tumor*** cytotoxicity. Together, these results suggest that ***arginase*** induction in macrophages can enhance ***tumor*** cell growth by providing them with polyamines and suppress ***tumor*** cytotoxicity by reducing NO production. It appears that L-arginine metabolism through the ***arginase*** and iNOS pathways in macrophages can have very different influences on the growth of nearby ***tumor*** cells depending on which pathway is prevailing.

L6 ANSWER 27 OF 43 USPATFULL on STN
ACCESSION NUMBER: 2000:1844 USPATFULL <<LOGINID::20060803>>
TITLE: Method of protein therapy by orally administering crosslinked protein crystals
INVENTOR(S): Navia, Manuel A., Lexington, MA, United States
St. Clair, Nancy L., Charlestown, MA, United States
PATENT ASSIGNEE(S): Vertex Pharmaceuticals, Inc., Cambridge, MA, United States (U.S. corporation)

NUMBER KIND DATE

PATENT INFORMATION: US 6011001 20000104
APPLICATION INFO.: US 1995-484978 19950607 (8)
RELATED APPLN. INFO.: Continuation of Ser. No. US 1993-17510, filed on 12 Feb 1993, now patented, Pat. No. US 5618710 which is a continuation-in-part of Ser. No. US 1992-864424, filed on 6 Apr 1992, now abandoned which is a continuation-in-part of Ser. No. US 1991-720237, filed on 24 Jun 1991, now abandoned which is a continuation-in-part of Ser. No. US 1990-562280, filed on 3 Aug 1990, now abandoned
DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Naff, David M.
LEGAL REPRESENTATIVE: Fish & Neave, Haley, Jr., James F., Pierri, Margaret A.
NUMBER OF CLAIMS: 4
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 19 Drawing Figure(s); 19 Drawing Page(s)
LINE COUNT: 3038
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A protein such as an enzyme or antibody is immobilized by crosslinking crystals of the protein with a multifunctional crosslinking agent. The crosslinked protein crystals may be lyophilized for storage. A preferred protein is an enzyme such as thermolysin, elastase, asparaginase, lysozyme, lipase or urease. Crosslinked enzyme crystals preferably retain at least 91% activity after incubation for three hours in the presence of a concentration of Pronase.TM. that causes the soluble uncrosslinked form of the enzyme to lose at least 94% of its initial activity under the same conditions. A preferred enzyme:Pronase.TM. ratio is 40:1. Enzyme crystals that are crosslinked may be microcrystals having a cross-section of 10.sup.-1 mm or less. Crosslinked enzyme or antibody crystals may be used in an assay, diagnostic kit or biosensor for detecting an analyte, in an extracorporeal device for altering a component of a fluid, in producing a product such as using crosslinked thermolysin crystals to produce aspartame and in separating a substance from a mixture. Enzyme or non-enzyme protein therapy can be performed by administering orally crosslinked enzyme crystals or crosslinked non-enzyme protein crystals that have a therapeutic affect. The crosslinked crystals have improved stability to proteases in the gut. Crosslinked lipase crystals may be administered for treatment where there is pancreatic insufficiency and/or fat malabsorption conditions in which lipase secretion is abnormally low.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 28 OF 43 USPATFULL on STN
ACCESSION NUMBER: 1999:166809 USPATFULL <<LOGINID::20060803>>
TITLE: Biosensors, extracorporeal devices and methods for detecting substances using crosslinked protein crystals
INVENTOR(S): Navia, Manuel A., Lexington, MA, United States
St. Clair, Nancy L., Charlestown, MA, United States
PATENT ASSIGNEE(S): Vertex Pharmaceuticals, Inc., Cambridge, MA, United States (U.S. corporation)

NUMBER KIND DATE

PATENT INFORMATION: US 6004768 19991221
APPLICATION INFO.: US 1995-484238 19950607 (8)
RELATED APPLN. INFO.: Continuation of Ser. No. US 1993-17510, filed on 12 Feb 1993, now patented, Pat. No. US 5618710 which is a continuation-in-part of Ser. No. US 1992-864424, filed on 6 Apr 1992, now abandoned which is a continuation-in-part of Ser. No. US 1991-720237, filed on 24 Jun 1991, now abandoned which is a continuation-in-part of Ser. No. US 1990-562280, filed on 3 Aug 1990, now abandoned
DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Naff, David M.
LEGAL REPRESENTATIVE: Fish & Neave, Haley, Jr., James F., Pierri, Margaret A.

NUMBER OF CLAIMS: 32
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 19 Drawing Figure(s); 19 Drawing Page(s)
LINE COUNT: 3066
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Proteins such as enzymes and antibodies are immobilized by crosslinking crystals of the proteins such as microcrystals having a cross-section of 10.sup.-1 mm or less with a multifunctional crosslinking agent. The crosslinked protein crystals may be lyophilized for storage. Crystals of an enzyme such as thermolysin, elastase, asparaginase, lysozyme, lipase or urease may be crosslinked to provide crosslinked enzyme crystals that retain at least 91% activity after incubation for three hours in the presence of a concentration of Pronase.TM. that causes the soluble uncrosslinked form of the enzyme to lose at least 94% of its initial activity under the same conditions. A preferred Pronase.TM.:enzyme ratio is 1:40. Crosslinked enzyme or antibody crystals may be used in an assay, diagnostic kit or biosensor for detecting an analyte, in an extracorporeal device for altering a component of a fluid, in producing a product such as using crosslinked thermolysin crystals to produce aspartame, in separating a substance from a mixture, and in therapy.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 29 OF 43 USPATFULL on STN
ACCESSION NUMBER: 1999:136679 USPATFULL <<LOGINID::20060803>>
TITLE: Methods of enzyme therapy by orally administering crosslinked enzyme crystals
INVENTOR(S): Navia, Manuel A., Lexington, MA, United States
St. Clair, Nancy L., Charlestown, MA, United States
PATENT ASSIGNEE(S): Vertex Pharmaceuticals, Inc., Cambridge, MA, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5976529		19991102
APPLICATION INFO.:	US 1995-477109		19950607 (8)

RELATED APPLN. INFO.: Continuation of Ser. No. US 1993-17510, filed on 12 Feb 1993, now patented, Pat. No. US 5618710 which is a continuation-in-part of Ser. No. US 1992-864424, filed on 6 Apr 1992, now abandoned which is a continuation-in-part of Ser. No. US 1991-720237, filed on 24 Jun 1991, now abandoned which is a continuation-in-part of Ser. No. US 1990-562280, filed on 3 Aug 1990, now abandoned
DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Naff, David M.
LEGAL REPRESENTATIVE: Fish & Neave, Haley, Jr., James F., Pierri, Margaret A.
NUMBER OF CLAIMS: 8
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 19 Drawing Figure(s); 19 Drawing Page(s)
LINE COUNT: 2922
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A protein such as an enzyme or antibody is immobilized by crosslinking crystals of the protein with a multifunctional crosslinking agent. The crosslinked protein crystals may be lyophilized for storage. A preferred protein is an enzyme such as thermolysin, elastase, asparaginase, lysozyme, lipase or urease. Crosslinked enzyme crystals preferably retain at least 91% activity after incubation for three hours in the presence of a concentration of Pronase.TM. that causes the soluble uncrosslinked form of the enzyme to lose at least 94% of its initial activity under the same conditions. A preferred enzyme:Pronase.TM. ratio is 1:40. Enzyme crystals that are crosslinked may be microcrystals having a cross-section of 10.sup.-1 mm or less. Crosslinked enzyme or antibody crystals may be used in an assay, diagnostic kit or biosensor for detecting an analyte, in an extracorporeal device for altering a component of a fluid, in producing a product such as using crosslinked thermolysin crystals to produce aspartame and in separating a substance from a mixture. Enzyme therapy such as lipase therapy can be performed by administering orally crosslinked lipase crystals.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 30 OF 43 Elsevier BIOBASE COPYRIGHT 2006 Elsevier Science B.V. on STN
ACCESSION NUMBER: 1999259495 ESBIOBASE <<LOGINID::20060803>>
TITLE: Cisplatin-mediated enzymatic changes in mice bearing ascites Dalton's lymphoma
AUTHOR: Prasad S.B.; Giri A.; Khynriam D.; Kharbangar A.; Nicol B.M.; Lotha C.
CORPORATE SOURCE: Dr. S.B. Prasad, Cell and Tumour Biology Laboratory, Department of Zoology, North-Eastern Hill University, Shillong-793 022, India.
SOURCE: Medical Science Research, (1999), 27/11 (723-730), 52 reference(s)
CODEN: MSCREJ ISSN: 0269-8951
DOCUMENT TYPE: Journal; Article
COUNTRY: United Kingdom
LANGUAGE: English
SUMMARY LANGUAGE: English
AB We have assayed the activities of nine enzymes in one or more tissues of normal, ***tumour*** (Dalton's lymphoma) bearing and cisplatin-***treated*** tumourous mice. In the ***liver*** of ***tumour*** -bearing hosts glucose-6-phosphatase and ***arginase*** activities decreased significantly while LDH activity increased significantly, as compared to normal mice. Cisplatin ***treatment*** for 24, 48, 72 and 96 h produced a significant rise in ***liver*** glucose-6-phosphatase and significant fall in LDH activity. LDH activity declined significantly in DL cells but increased significantly in ascites supernatants following cisplatin ***treatment*** which may suggest the leakage/release of LDH from the DL cells. The serum and ascites supernatant glucose levels increased significantly following cisplatin ***treatment*** . ***Arginase*** activity rose significantly from 8 to 48 h of cisplatin ***treatment*** but fell later at 96 h. The raised activity of cathepsin B and H in the ascites supernatants and sera after cisplatin ***treatment*** may suggest their secretion in the fluids from different tissues and may also contribute to the tumouricidal effect mediated by cisplatin. As compared to normal animals serum GOT and GPT activities were significantly higher in ***tumour*** -bearing hosts. Cisplatin ***treatment*** led to a significant increase in GOT activity in ascites supernatants. Activities of membrane enzymes, (Na.sup.+ + K.sup.+) Mg.sup.2.sup.+-ATPase as well as 5'-nucleotidase, gradually declined in the ***tumour*** cells, with concomitant rises in ascites supernatants, following cisplatin ***treatment*** . The lower activities could be due to loss from the cells or some other mechanism and may also add indirectly to the antitumour activity of cisplatin. As compared to normal mice, the total protein concentration in the kidney of ***tumour*** -bearing hosts decreased significantly but increased significantly in serum. Cisplatin ***treatment*** resulted in significant decreases in protein concentration in kidney, serum and DL cells. It is suggested that these enzymatic changes may affect the metabolism of ***tumour*** cells and other tissues, perhaps facilitating the anticancer activity of cisplatin.

L6 ANSWER 31 OF 43 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1998:204297 CAPLUS <<LOGINID::20060803>>
DOCUMENT NUMBER: 128:240349
TITLE: Use of a non-mammalian DNA virus to express an exogenous gene in a mammalian cell for gene therapy in treatment of gene deficiency disorder or liver cancer
INVENTOR(S): Boyce, Frederick M.
PATENT ASSIGNEE(S): General Hospital Corp., USA
SOURCE: U.S., 25 pp., Cont.-in-part of U.S. 311,157.
CODEN: USXXAM
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE

US 5731182	A	19980324	US 1995-486341	19950607
US 5871986	A	19990216	US 1994-311157	19940923
CA 2200835	AA	19960328	CA 1995-2200835	19950908
WO 9609074	A1	19960328	WO 1995-US11456	19950908

W: AM, AT, AU, BB, BG, BR, BY, CA, CH, CN, CZ, DE, DK, EE, ES, FI, GB, GE, HU, IS, JP, KE, KG, KP, KR, KZ, LK, LR, LT, LU, LV, MD, MG, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, TJ, TM, TT

RW: KE, MW, SD, SZ, UG, AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG

AU 9536750	A1	19960409	AU 1995-36750	19950908
AU 702830	B2	19990304		
EP 785803	A1	19970730	EP 1995-934407	19950908

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE

CN 1172435	A	19980204	CN 1995-196379	19950908
JP 10506530	T2	19980630	JP 1995-510940	19950908
ZA 9507797	A	19960708	ZA 1995-7797	19950915
TW 464500	B	20011121	TW 1995-84109934	19950923
US 6238914	B1	20010529	US 1996-752030	19961119

PRIORITY APPLN. INFO.: US 1994-311157 A2 19940923
US 1995-486341 A 19950607
WO 1995-US11456 W 19950908

AB Disclosed is a method of expressing an exogenous gene in a mammalian cell, involving infecting the cell with a non-mammalian virus, such as a baculovirus, whose genome carries an exogenous gene, and growing the cell under conditions such that the gene is expressed. Exogenous genes are delivered to mammalian cells by use of a transfer vector such as that described in the figure. Also disclosed is a method of treating a gene deficiency disorder in a mammal by providing to a cell a therapeutically effective amt. of a virus whose genome carries an exogenous gene and growing the cell under conditions such that the exogenous gene is expressed in the mammal.

REFERENCE COUNT: 14 THERE ARE 14 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 32 OF 43 USPATFULL on STN
ACCESSION NUMBER: 1998:156918 USPATFULL <<LOGINID::20060803>>
TITLE: Crosslinked protein crystals
INVENTOR(S): Navia, Manuel A., Lexington, MA, United States
St. Clair, Nancy L., Charlestown, MA, United States
PATENT ASSIGNEE(S): Vertex Pharmaceuticals, Inc., Cambridge, MA, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5849296		19981215
APPLICATION INFO.:	US 1995-476267		19950607 (8)

RELATED APPLN. INFO.: Continuation of Ser. No. US 1993-17510, filed on 12 Feb 1993, now patented, Pat. No. US 5618710 which is a continuation-in-part of Ser. No. US 1992-864424, filed on 6 Apr 1992, now abandoned which is a continuation-in-part of Ser. No. US 1991-720237, filed on 24 Jun 1991, now abandoned which is a continuation-in-part of Ser. No. US 1990-562280, filed on 3 Aug 1990, now abandoned
DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Naff, David M.
LEGAL REPRESENTATIVE: Fish & Neave, Haley, Jr., James F., Pierri, Margaret A.
NUMBER OF CLAIMS: 15
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 19 Drawing Figure(s); 19 Drawing Page(s)
LINE COUNT: 3122
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB A protein such as an enzyme or antibody is immobilized by crosslinking crystals of the protein with a multifunctional crosslinking agent. The crosslinked protein crystals may be lyophilized for storage. A preferred protein is an enzyme such as thermolysin, elastase, asparaginase,

lysozyme, lipase or urease. Crosslinked enzyme crystals preferably retain at least 91% activity after incubation for three hours in the presence of a concentration of Pronase.TM. that causes the soluble uncrosslinked form of the enzyme to lose at least 94% of its initial activity under the same conditions. A preferred enzyme:Pronase.TM. ratio is 1:40. Enzyme crystals that are crosslinked may be microcrystals having a cross-section of 10.sup.-1 mm or less. Crosslinked enzyme or antibody crystals may be used in an assay, diagnostic kit or biosensor for detecting an analyte, in an extracorporeal device for altering a component of a fluid, in producing a product such as using crosslinked thermolysin crystals to produce aspartame, in separating a substance from a mixture, and in therapy.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 33 OF 43 USPATFULL on STN
ACCESSION NUMBER: 97:29369 USPATFULL <<LOGINID::20060803>>
TITLE: Crosslinked enzyme crystals
INVENTOR(S): Navia, Manuel A., Lexington, MA, United States
St. Clair, Nancy L., Charlestown, MA, United States
PATENT ASSIGNEE(S): Vertex Pharmaceuticals, Inc., Cambridge, MA, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5618710		19970408
APPLICATION INFO.:	US 1993-17510		19930212 (8)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 1992-864424, filed on 6 Apr 1992, now abandoned which is a continuation-in-part of Ser. No. US 1991-720237, filed on 24 Jun 1991, now abandoned which is a continuation-in-part of Ser. No. US 1990-562280, filed on 3 Aug 1990, now abandoned
DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Naff, David M.
LEGAL REPRESENTATIVE: Fish & Neave, Haley, Jr., James F., Pierri, Margaret A.
NUMBER OF CLAIMS: 13
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 19 Drawing Figure(s); 19 Drawing Page(s)
LINE COUNT: 3106
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A protein such as an enzyme of antibody is immobilized by crosslinking crystals of the protein with a multifunctional crosslinking agent. The crosslinked protein crystals may be lyophilized for storage. A preferred protein is an enzyme such as thermolysin, elastase, asparaginase, lysozyme, lipase or urease. Crosslinked enzyme crystals preferably retain at least 91% activity after incubation for three hours in the presence of a concentration of Pronase.TM. that causes the soluble uncrosslinked form of the enzyme to lose at least 94% of its initial activity under the same conditions. A preferred enzyme:Pronase.TM. ratio is 1:40. Enzyme crystals that are crosslinked may be microcrystals having a cross-section of 10.sup.-1 mm or less. Crosslinked enzyme or antibody crystals may be used in an assay, diagnostic kit or biosensor for detecting an analyte, in an extracorporeal device for altering a component of a fluid, in producing a product such as using crosslinked thermolysin crystals to produce aspartame, in separating a substance from a mixture, and in therapy.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 34 OF 43 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1996:363519 CAPLUS <<LOGINID::20060803>>
DOCUMENT NUMBER: 125:27687
TITLE: Use of a non-mammalian DNA virus to express an exogenous gene in a mammalian cell for gene therapy in treatment of gene deficiency disorder or liver cancer
INVENTOR(S): Boyce, Frederick M.
PATENT ASSIGNEE(S): General Hospital Corporation, USA
SOURCE: PCT Int. Appl., 77 pp.

CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9609074	A1	19960328	WO 1995-US11456	19950908
W: AM, AT, AU, BB, BG, BR, BY, CA, CH, CN, CZ, DE, DK, EE, ES, FI, GB, GE, HU, IS, JP, KE, KG, KP, KR, KZ, LK, LR, LT, LU, LV, MD, MG, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, TJ, TM, TT				
RW: KE, MW, SD, SZ, UG, AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
US 5871986	A	19990216	US 1994-311157	19940923
US 5731182	A	19980324	US 1995-486341	19950607
AU 9536750	A1	19960409	AU 1995-36750	19950908
AU 702830	B2	19990304		
EP 785803	A1	19970730	EP 1995-934407	19950908
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE				
JP 10506530	T2	19980630	JP 1995-510940	19950908

PRIORITY APPLN. INFO.: US 1994-311157 A 19940923
US 1995-486341 A 19950607
WO 1995-US11456 W 19950908

AB Disclosed is a method of expressing an exogenous gene in a mammalian cell, involving infecting the cell with a non-mammalian virus, such as a baculovirus, whose genome carries an exogenous gene, and growing the cell under conditions such that the gene is expressed. Exogenous genes are delivered to mammalian cells by use of a transfer vector such as that described in the figure. Also disclosed is a method of treating a gene deficiency disorder in a mammal by providing to a cell a therapeutically effective amt. of a virus whose genome carries an exogenous gene and growing the cell under conditions such that the exogenous gene is expressed in the mammal.

L6 ANSWER 35 OF 43 BIOTECHNO COPYRIGHT 2006 Elsevier Science B.V. on STN
ACCESSION NUMBER: 1990:20194091 BIOTECHNO <<LOGINID::20060803>>
TITLE: Inhibition by mouse .alpha./.beta.-interferon of the multiplication of .alpha./.beta.-interferon-resistant Friend erythroleukemia cells cocultured with mouse hepatocytes
AUTHOR: Yasui H.; Proietti E.; Vignaux F.; Eid P.; Gresser I.
CORPORATE SOURCE: Laboratory of Viral Oncology (UPR CNRS 274), Groupe de Laboratoires de l'Institut de Recherches Scientifiques sur le Cancer, 7 Rue Guy Moquet, 94801 Villejuif Cedex, France.
SOURCE: Cancer Research, (1990), 50/12 (3533-3539)
CODEN: CNREA8 ISSN: 0008-5472
DOCUMENT TYPE: Journal; Article
COUNTRY: United States
LANGUAGE: English
SUMMARY LANGUAGE: English
AN 1990:20194091 BIOTECHNO <<LOGINID::20060803>>
AB Administration of .alpha./.beta.-interferon (IFN) exerts a marked antitumor effect in DBA/2 mice given injections i.v. of large numbers of IFN-.alpha./.beta.-resistant erythroleukemia cells (FLC). To investigate the possible mechanisms of FLC ***tumor*** inhibition in the ***liver*** of interferon- ***treated*** mice, we developed an in vitro model consisting of a coculture of IFN-.alpha./.beta.-resistant 3C18 FLC and syngeneic mouse hepatocytes. Whereas IFN-.alpha./.beta. did not inhibit the multiplication of 3C18 FLC cultivated alone, it effectively inhibited the multiplication of 3C18 FLC in coculture with hepatocytes. The inhibitory effect was directly proportional to the amount of IFN-.alpha./.beta. added to the cocultures, and more than 90% inhibition of FLC multiplication was noted with 1.6 x 10.sup.5 IU/ml of IFN-.alpha./.beta. on Day 3 of coculture. When FLC were separated from the monolayer of hepatocytes by a pored membrane (0.4 .mu.m), the inhibitory effect on FLC proliferation was unchanged, indicating that a

soluble factor(s) released from IFN- ***treated*** hepatocytes was most important in the inhibition of FLC multiplication. An inhibitory activity of FLC multiplication was detected only in the conditioned medium of IFN- ***treated*** hepatocytes but not in the conditioned medium of control hepatocytes nor in extracts of IFN- ***treated*** or control hepatocytes. The inhibitory factor(s) in the conditioned medium of IFN- ***treated*** hepatocytes was retained by an ultrafiltration membrane (M(r) cut off 10,000) and its activity was completely abrogated by trypsin digestion. Its stability to ***treatment*** with 1 M acetic acid as well as lack of correlation between the antiproliferative effect and the amount of L-arginine in the medium distinguished this factor(s) from ***liver*** ***arginase*** which was also found to be a potent inhibitor of FLC multiplication in vitro. The inhibitory factor(s) was also distinguishable in its biological activity from IFN.gamma., interleukin 1.alpha. and .beta., and transforming growth factor .beta.1 and .beta.2. These results suggest the possibility that the inhibitory effect of IFN-.alpha./.beta. on the development of 3C18 FLC in the livers of IFN- ***treated*** mice may be mediated by an IFN-induced inhibitor of FLC multiplication.

L6 ANSWER 36 OF 43 PASCAL COPYRIGHT 2006 INIST-CNRS. ALL RIGHTS RESERVED.
on STN DUPLICATE 4
ACCESSION NUMBER: 1990-0305952 PASCAL <<LOGINID::20060803>>
TITLE (IN ENGLISH): Properties of arginase immobilized in a fibrin clot
AUTHOR: DIEZ A.; CAMPO M. L.; SOLER G.
CORPORATE SOURCE: Univ. extremadura, fac. veterinaria, dep. bioquimica biologia molecular genetica, Caceres, Spain
SOURCE: Biotechnology and applied biochemistry, (1990), 12(3), 237-244, 26 refs.
ISSN: 0885-4513 CODEN: BABIEC
DOCUMENT TYPE: Journal
BIBLIOGRAPHIC LEVEL: Analytic
COUNTRY: United States
LANGUAGE: English
AVAILABILITY: INIST-17979, 354000008999800020
AN 1990-0305952 PASCAL <<LOGINID::20060803>>
AB Rat ***liver*** ***arginase*** was covalently trapped in a fibrin clot. Among the physicochemical properties of the enzyme studied were Mn.sup.2.sup.+ requirement, pH behavior, temperature and time stability, effect of denaturing agents, and kinetic properties. The properties so far examined may enhance the use of immobilized ***arginase*** in ***cancer*** ***therapy***

L6 ANSWER 37 OF 43 CAPLUS COPYRIGHT 2006 ACS on STN DUPLICATE 5
ACCESSION NUMBER: 1988:69153 CAPLUS <<LOGINID::20060803>>
DOCUMENT NUMBER: 108:69153
TITLE: Effects of female sex steroids on concanavalin A-mediated agglutination of hepatocytes from nonregenerating and regenerating rat liver and hepatic tumor marker enzymes
AUTHOR(S): Annapurna, V. V.; Mukundan, M. A.; Sesikeran, B.; Bamji, Mahtab S.
CORPORATE SOURCE: Indian Counc. Med. Res., Natl. Inst. Nutr., Hyderabad, 500 007, India
SOURCE: Biochemical Medicine and Metabolic Biology (1987), 38(3), 259-64
CODEN: BMMBES; ISSN: 0885-4505
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Effect of treatment of female rats with an oral contraceptive agent (OCA), Ovulen-50, for 7 wk on agglutination of hepatocytes with Con A and activities of certain tumor marker enzymes were examd. to det if OCA treatment is related to preneoplastic or neoplastic processes. Hepatocytes from regenerating and nonregenerating livers of control female rats showed negligible agglutination with Con A, whereas hepatocytes from nonregenerating, but not from the regenerating, livers of female rats treated with a combination of 5 .mu.g ethinylestradiol and 100 .mu.g ethynodiol diacetate showed agglutination. Of the ***tumor*** marker enzymes: hepatic glucose 6-phosphatase, .gamma.-glutamyl transpeptidase (.gamma.-GT), and ***arginase*** , examd. in the ***liver*** , only

.gamma.-GT showed an increase in activity in the steroid- ***treated*** rats. Plasma alk. phosphatase activity was also higher in the treated animals. However, the magnitude of the changes obsd. was relatively small and perhaps unrelated to the neoplastic process.

L6 ANSWER 38 OF 43 CAPLUS COPYRIGHT 2006 ACS on STN DUPLICATE 6
ACCESSION NUMBER: 1984:622236 CAPLUS <<LOGINID::20060803>>
DOCUMENT NUMBER: 101:222236
TITLE: ***Cancer*** ***therapy*** with chemically modified enzymes. II. The ***therapeutic*** effectiveness of ***arginase*** , and ***arginase*** modified by the covalent attachment of polyethylene glycol, on the Taper ***liver*** ***tumor*** and the L5178Y murine leukemia
AUTHOR(S): Savoca, K. V.; Davis, F. F.; Van Es, T.; McCoy, J. R.; Palczuk, N. C.
CORPORATE SOURCE: Dep. Biol., Rutgers Univ., New Brunswick, NJ, 08903, USA
SOURCE: Cancer Biochemistry Biophysics (1984), 7(3), 261-8
CODEN: CABCD4; ISSN: 0305-7232
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Monomethoxypolyethylene glycol (PEG) was attached covalently to arginase; PEG-arginase was effective in prolonging the survival times of mice injected with the Taper liver tumor, whereas unmodified arginase was ineffective. PEG-arginase was more effective than arginase in the in vitro destruction of L5178Y mouse leukemia. However, neither PEG-arginase nor arginase inhibited the in vivo growth of this tumor.

L6 ANSWER 39 OF 43 BIOTECHNO COPYRIGHT 2006 Elsevier Science B.V. on STN
ACCESSION NUMBER: 1981:11071798 BIOTECHNO <<LOGINID::20060803>>
TITLE: Regulation of urea cycle enzymes in transplantable hepatomas and in the livers of tumor-bearing rats and humans
AUTHOR: Brebnor L.D.; Grimm J.; Balinsky J.B.
CORPORATE SOURCE: Dept. Biochem., Univ. Witwatersrand, Johannesburg 2001, South Africa.
SOURCE: Cancer Research, (1981), 41/7 (2692-2699)
CODEN: CNREA8
DOCUMENT TYPE: Journal; Article
COUNTRY: United States
LANGUAGE: English
AN 1981:11071798 BIOTECHNO <<LOGINID::20060803>>
AB The levels of the five enzymes of the urea cycle were measured in normal 5-week-old rats, in a transplantable hepatoma, and in the livers of ***tumor*** -bearing rats (host livers). The levels of all five enzymes were much lower in the hepatoma, although there was no exact correlation of the decrease in levels. In host livers, the levels were higher than in the tumors, but lower than in normal ***liver*** . The levels of all five urea cycle enzymes were positively correlated with dietary protein content in normal livers, in hepatomas, and in host livers. In fact, the hepatomas showed the greatest changes in response to diet. On all diets, the levels in host ***liver*** remained below those in normal ***liver*** , indicating that the decreased level was probably not due to preferential utilization of nutrients by the ***tumor*** . The levels of urea cycle enzymes in normal ***liver*** were not altered by a single injection of glucocorticoid, glucagon, or dibutyryl cyclic adenosine 3':5'-monophosphate. By contrast, in hepatoma, the levels were usually significantly elevated by the same ***treatment*** . In addition, the levels in host livers were always significantly elevated and were usually above those in normal animals, whether the latter were hormone ***treated*** or not. Injection of plasma from ***tumor*** -bearing rats into normal animals produced a decrease in the levels of all five enzymes; if glucagon was injected together with the plasma, large increases in levels were observed. This result supports the concept of a humoral factor produced by the ***tumor*** which affects the levels and the inducibility of urea cycle enzymes in host livers. Autopsied human primary hepatomas also showed levels of urea cycle enzymes below those in normal livers with host livers having intermediate values. A cell line derived from a human hepatoma showed induction of

arginase by glucocorticoid in culture; in this, it resembled a cell line of the rat hepatoma. Tyrosine aminotransferase in human hepatoma cells was not induced by glucocorticoid; in this, it differed from the rat hepatoma cells where induction of this enzyme was observed.

L6 ANSWER 40 OF 43 CAPLUS COPYRIGHT 2006 ACS on STN DUPLICATE 7
ACCESSION NUMBER: 1976:590417 CAPLUS <<LOGINID::20060803>>
DOCUMENT NUMBER: 85:190417
TITLE: Enzyme studies on normal liver and primary hepatoma subjected to dietary and hormonal stimuli
AUTHOR(S): D'Souza, R. A.; Bhide, S. V.
CORPORATE SOURCE: Tata Mem. Cent., Cancer Res. Inst., Bombay, India
SOURCE: Indian Journal of Experimental Biology (1976), 14(4), 415-17
CODEN: IJEBA6; ISSN: 0019-5189
DOCUMENT TYPE: Journal
LANGUAGE: English
AB In an attempt to study metabolic regulation in normal liver and hepatoma, normal mice and mice bearing primary liver tumors were subjected to various stimuli such as feeding with 10 and 20% casein and i.p. administration of hydrocortisone acetate. Untreated normal and tumor bearing livers were used for comparison. Activities of ornithine carbamoyl transferase, aspartate carbamoyl transferase, ***arginase*** , glucose-6-phosphatase, and fructose-1,6-diphosphatase were measured in untreated, and ***treated*** , normal ***liver*** and ***tumor*** tissue. The responses of the enzymes to cortisone treatment did not reveal any specific pattern characteristic of the hepatoma. However feeding of 10 and 20% casein produced differential responses in both the tissues. Unlike normal liver, hepatoma failed to produce any significant alteration in the activities of aspartate and ornithine carbamoyl transferases.

L6 ANSWER 41 OF 43 CAPLUS COPYRIGHT 2006 ACS on STN DUPLICATE 8
ACCESSION NUMBER: 1973:67940 CAPLUS <<LOGINID::20060803>>
DOCUMENT NUMBER: 78:67940
TITLE: Differential metabolic responses of liver tissue of rats and mice to thioacetamide treatment
AUTHOR(S): Bhide, S. V.
CORPORATE SOURCE: Biol. Div., Cancer Res. Inst., Bombay, India
SOURCE: Indian Journal of Cancer (1972), 9(2), 154-9
CODEN: IJCAAR; ISSN: 0019-509X
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Thioacetamide [62-55-5] administered to mice and rats increased the activity of aspartate carbamoyltransferase [9012-49-1] in the liver. Thioacetamide decreased glycogen [9005-79-2] content in the liver of 6-month-old mice and of 19-month-old rats. On the other hand, lactic acid [50-21-5] content was increased in 13-month-old mice and 19-mouth-old rats. Activities of ornithine carbamoyltransferase [9001-69-8], ***arginase*** [9000-96-8], and xanthine oxidase [9002-17-9] were altered only in the ***liver*** of ***treated*** mice undergoing ***malignant*** transformation. Activities of glucose 6-phosphatase [9001-39-2] and fructose-1,6-diphosphatase [9001-52-9] were decreased in the liver of both animals.

L6 ANSWER 42 OF 43 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1970:53515 CAPLUS <<LOGINID::20060803>>
DOCUMENT NUMBER: 72:53515
TITLE: Effect of an alkylating agent on liver arginase in normal and Ehrlich carcinoma-bearing mice
AUTHOR(S): Abreu, R. Raposo; Abreu, Luiz A.
CORPORATE SOURCE: Inst. Oswaldo Cruz, Rio de Janeiro, Brazil
SOURCE: Hospital (Rio de Janeiro) (1969), 76(1), 347-51
CODEN: HOSOA3; ISSN: 0018-5469
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The arginase activity of the liver of intact mice was not changed by i.p. injection of 200 mg/kg cyclophosphamide (I). Significantly increased arginase activity of the liver was observed 12 and 14 days after i.p. transplantation of Ehrlich ascites carcinoma. In the latter group the

activity was further increased by I, and the effect set in within 3 hr after i.p. injection.

L6 ANSWER 43 OF 43 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1951:30390 CAPLUS <<LOGINID::20060803>>
DOCUMENT NUMBER: 45:30390
ORIGINAL REFERENCE NO.: 45:5290f-h
TITLE: Enzyme content of benign and malignant liver tumors. I. Arginase and histidase
AUTHOR(S): Viollier, G.
CORPORATE SOURCE: Med. Univ., Basel, Switz.
SOURCE: Verhandl. schweiz. Ver. Physiol. u. Pharmakol. (1950) C34-6,C37-9
From: Helv. Physiol. et Pharmacol. Acta 8, No. 2
DOCUMENT TYPE: Journal
LANGUAGE: Unavailable
AB Rats kept 20-50 weeks on a low-choline diet developed benign hepatic tumors, and rats fed dimethylaminoazobenzene (I) developed malignant hepatic tumors. Extrahepatic malignant tumors were developed in rats by subcutaneous injection of benzopyrene or methylcholanthrene. The av. ***arginase*** activity per unit protein was about half the control value in the ***tumor*** -free ***liver*** tissue of the I-***treated*** animals which had developed tumors, and about 1/6 of the control value in the tumors of these animals. A marked change in arginase activity was not seen in the other exptl. groups, including I-treated animals which did not develop tumors. A slight rise in this activity in livers from choline-deficient animals was probably due to the high sulfhydryl content of their diet. The av. histidase activity per unit protein was lower in all the exptl. groups, as compared with their respective control groups, but was especially low in the I-treated animals, and was negligible in the hepatic tumors of these animals.

=> d his

L1 QUE (ARGINASE OR CANAVANASE OR (ARGININE ADJ AMIDINASE))

L2 20460 S L1
L3 1236 S (MALIGNAN? OR CANCER OR TUMOR OR TUMOUR OR NEOPLAS?)(S)L2
L4 281 S (TREAT? OR PREVENT? OR MODULAT? OR THERAP?)(S)L3
L5 69 S LIVER (S)L4
L6 43 DUP REM L5 (26 DUPLICATES REMOVED)

=> log y